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SYMPOSIUM

Leucosis and Respiratory Disease in Poultry

A symposium, having the title above, was heard by the Association of Veterinary Teachers and Research Workers at a meeting held on January 6th this year. The speakers were: Mr. P. M. Biggs, Dr. J. G. Campbell, Mr. J. D. Blaxland, Dr. A. S. King, Dr. R. F. Gordon, and Mr. F. T. W. Jordan. The first three papers, comprising the morning session of the symposium, are published below; the remainder—on the subject of respiratory disease—will be published next week.

Lymphoid and Haemopoietic Tissue

BY

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THE problem of the distribution of haemopoietic, and particularly of lymphoid tissue in the domestic chicken has recently been approached chiefly by pathologists primarily interested in the problem of the fowl leucosis complex. To-day I hope to try to reverse this procedure by a consideration of the position from an anatomical standpoint, bearing in mind its relevance to this group of conditions. The long incubation period and possible latent infection with certain of the manifestations of the leucosis complex make it impossible to be emphatic about where the normal ends and abnormal begins. One can only describe what is seen in the apparently normal and certainly clinically healthy bird.

In the short time available it is only possible to concern ourselves, in the most part, with generalities. Details I have confined to the domestic chicken unless stated otherwise.

In the chicken the formation of red cells and thrombocytes takes place intravascularly (that is within the sinusoids of haemopoietic tissue) and granular leucocytes extravascularly. It is also as well to remember that there is good evidence (Jordan, 1936) for the large lymphocyte being the common stem cell of all blood cells.

Considering first the embryo we find that in the domestic chicken the yolk sac is the main haemopoietic organ in the early stages. According to Sandreuter (1951) the liver and spleen have a slight erythropoietic activity in the domestic chicken between the seventh and ninth days, but both these organs are more concerned with the production of lymphocytes and granulocytes. The liver, and to a greater extent the spleen, are the main granulocytopoietic organs in the latter part of embryonic life. This function, together with erythropoiesis and lymphopoiesis, is taken up by the bone marrow which, by the time of hatching, is the only organ with erythropoietic activity and also the primary source of granular leucocytes. A few days before hatching definitive lymphocytes are seen when the liver, spleen, and bone marrow take up a lymphopoietic function which is continued in the post-embryonal period. Apart from these specific organs lymphopoiesis particularly and some granulocyte and erythrocyte production occurs in the thymus, bursa of Fabricius, and mesenchymal connective tissue throughout the embryo body.

In the post-embryonal period and the adult bird we find that erythropoiesis and the greater part of granulocyte formation occurs in the red bone marrow. This tissue is found in most bones in the very young but is replaced by extensions of air sacs comparatively early in some (*e.g.* humerus), and in others haemopoiesis ceases or is reduced in the adult (*e.g.* radius and ulna).

Lastly, the subject which most interests us to-day, the post-embryonal and adult distribution of lymphoid tissue. Lymph nodes as seen in mammals are absent in birds. Two pairs of structures termed lymph nodes, but differing in structure from the mammalian node, are found only in some water, sea, and shore birds (*e.g.* goose and duck). The accepted sites for this tissue in the chicken are: spleen, thymus, bursa of Fabricius, and intestinal tract. Apart from these there are other sites where lymphoid tissue is con-

stantly present. The first, and probably the most important from the anatomical point of view, was first described by a group of workers in Japan (Kihara & Naito, 1933, and Kondo, 1937a, b, c) in the 1930s. They described lymph nodules placed irregularly on the course of the lymph vessels of many avian species, the domestic chicken being investigated in detail. This is of particular interest because lymphoid tissue has been thought to be associated with lymph vessels only in some water, sea, and shore birds as the two pairs of lymph nodes mentioned before. I have spent some time investigating these lymph nodules in *Gallus domesticus* and have found them to vary from small infiltrations of the vessel wall and surrounding connective tissue to nodules up to 2.5 mm. long (Biggs, 1956). They are placed irregularly along the lymphatics and appear to be more abundant in the hind limb. Microscopically they are unencapsulated condensations of lymphoid tissue. According to Kondo they start to develop 28 days after hatching and by the 42nd day they contain secondary nodules. In the larger nodules trabeculae originating from the vessel wall may be seen penetrating the nodule. I have seen no lymph sinuses or vessels connecting the lumen of the lymph vessel with the substance of the nodule. But the nodules do, however, have a plexus of small blood vessels which are often filled with lymphocytes. This indicates a lymphopoietic activity which may answer in part the riddle of the source of blood lymphocytes. In young birds areas of myelocytes are often seen in these nodules. They may, therefore, in common with lymphoid tissue elsewhere in the avian body, have a granulocytopoietic function.

Apart from these fairly well-organised bodies, lymphoid tissue is found to be very widespread in the avian body. Lymph nodules and areas have been described in connective tissue (Danchakoff, 1915-16 and Mjassojedoff, 1926) and often in association with blood vessels. Mjassojedoff described the presence of lymphoid tissue in every organ where connective tissue is present, *e.g.* lung, liver, kidney, gonads, etc. I have personally seen it in apparently normal chickens in trachea, lungs, kidneys, ureters, bone marrow, pancreas, adrenal, gonads, thyroids, pituitary, liver, and connective tissue. In the liver much of this tissue is associated with the portal vessels but as with other organs it is not uncommon to find areas in the parenchyma. I have so far only made a detailed study of adrenals, 38 of which have been examined by serial section from six-week-old Rhode Island Reds. In every one some lymphoid tissue was present. The volume varied considerably, something like 200-fold, that is from about .01 per cent. to 2.0 per cent. of the whole organ. As these areas are typical of what has been termed " ectopic " lymphoid areas by Lucas and co-workers (1949 and 1954) in America, I think it is excusable to describe them in a little detail as seen in the adrenals of the six-week-old birds. They may appear anywhere in the organ but often are associated with the connective tissue capsule or blood vessels. Some areas may have a very close association with a blood vessel at times even protruding into its lumen. They may vary in size from an accumulation of a few cells to areas as large as $\frac{3}{4}$ mm. in diameter. At this age only occasional secondary nodules were seen, though they have been described elsewhere in older birds for this type of tissue. Some may be discrete or as is most often the case they may infiltrate the surrounding tissue. Adrenal cells, both " cortical " and " medullary ", may be surrounded and engulfed by the advancing lymphocytes but apparently are unaffected there being no indications of necrobiosis or cell death. This is contrary to that reported by Lucas and co-workers for the pancreas. In my young birds myelocytes were also sometimes present.

To summarise the histological characteristics of lymphoid tissue in the chicken one finds that: it is intimately related to the surrounding tissue, a connective tissue capsule being absent; though the small lymphocyte predominates, larger forms are seen in greater numbers than in mammalian lymphoid tissue; secondary nodules are surrounded by a delicate connective tissue capsule and usually contain larger and more closely packed lymphocytes.

One might now ask oneself what is the significance of the facts? 1. With the absence of a reticulo-endothelial network traversing the lymphatic system as seen in the mammalian node there is no filtering action in this system. 2. There is certainly ample lymphoid tissue for the lymphocyte requirements of the blood. 3. The close association between lymphoid tissue and its surrounding tissue means care must be taken in interpreting the infiltration of this surrounding tissue by the lymphocytes as a pathological process. 4. Lastly can one say anything about the significance of the so-called " ectopic " areas? Are they (a) normal, (b) normal areas which may be affected by lymphomatosis, or (c) expressions of a latent form of lymphoid disease? I submit that, at the least, this is a problem that cannot be answered with certainty as yet. But these areas are present in large numbers of clinically healthy birds, in fact with their widespread distribution it is possible that they are present in every domestic chicken. This taken with evidence of changes occurring in these areas after experimental infection with various organisms leads one to think that it is reasonable to look on the so-called ectopic lymphoid areas as a normal expression of lymphoid tissue which may become hyperplastic or even neoplastic at different stages of lymphoid disease.

References

BIGGS, P. M. (1956). *Acta. Anat.*, in press.

DANCHAKOFF, V. (1915-16). *Anat. Rec.*, **10.** 483-92.

JORDAN, H. E. (1936). *Amer. J. Anat.* **59.** 249-83.

KIHARA, T., & NAITO, E. (1933). *Folia Anat. Jap.* **11.** 405-13.

KONDO, M. (1937a). *Ohajimas Folia Anat. Jap.* **15.** 309-25.

———, (1937b). *Ibid.* **15.** 329-48.

———. (1937c). *Ibid.* **15.** 349-55.

LUCAS, A. M. (1949). *Amer. J. Path.* **25.** 1197-1213.

———, DENINGTON, E. M., COTTRAL, G. E., & BURMESTER, B. R. (1954) *Poult. Sci.* **33.** 562-70. *Ibid.* **33.** 571-84.

MJASSOJEDOFF S. W. (1926). *Folia Haemat.* (Lpz.) **32.** 263-96.

SANDREUTER, A. (1951). *Acta Anat.* **11.** Suppl. 14.

Leucosis and Fowl Paralysis Compared and Contrasted

BY

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THE leucotic complex of diseases in the fowl has long been the subject of controversy among pathologists, who find themselves in acute difference over the question of the inclusion of fowl paralysis within the complex. A brief glance at the history of these diseases will help to understand how this divergence of opinion has arisen. When fowl paralysis was first recorded in Britain by Galloway (1929) it was described as a disease entity characterised primarily by paralytic symptoms in immature fowls associated with typical infiltrations of various peripheral nerves by cells of the lymphoid series. This was in accord with reports of the disease from various parts of the world, and the name neurolymphomatosis gallinarum was proposed and generally adopted.

It was known from the outset that nerve lesions were not the only manifestation, and the terms visceral lymphomatosis and ocular lymphomatosis were given to the other forms.

It is as well to realise that long before the recognition of fowl paralysis in its various guises, the conditions now known as the leucoses were familiar to pathologists. These conditions, now classified as lymphoid leucosis, myeloid leucosis and erythroleucosis, were not found to be associated with nerve lesions, although later studies have shown that on rare occasions an invasion of nerves with cells of these different series may occur. In view of the undoubted malignancy of the various leucoses, this is not surprising.

The form of fowl paralysis called visceral lymphomatosis bears some gross resemblance to the discrete form of lymphoid leucosis, and it is this similarity which caused various workers, especially in the United States of America, to include all forms of fowl paralysis in the leucotic complex. Because of this, visceral lymphomatosis and lymphoid leucosis were believed by some to be synonymous, and by implication had a common aetiology. Many workers on this side of the Atlantic accepted this as a working classification, tending to overlook certain discrepancies. Indeed, it must be admitted that there was no great harm apparent in this from the viewpoint of the poultry farmer and the busy diagnostic laboratories. But it was obvious to some that this classification meant that lymphoid leucosis, whether discrete (lymphocytoma) or diffuse (lymphosarcoma), was synonymous with not only the visceral form of fowl paralysis, but with the neural and ocular forms also, none of these being neoplastic conditions. Not only this, but the other forms of leucosis were also included, and a tendency became evident to report all these conditions simply as fowl paralysis.

The situation was not clarified as research into the aetiology of these various conditions continued, and it became apparent that myeloid and erythroleucosis were associated with a closely similar if not identical filterable agent or "virus," comparable, but not apparently identical with the "virus" Rous fibromyxosarcoma of fowls, so well-known to cancer research workers all over the world. Lymphoid leucosis, on the other hand, has not been shown to have an associated filterable agent except by Burmester and his colleagues, and some doubt has been raised in the minds of a few British workers that the Burmester transmissible lymphoid tumour is in fact comparable with lymphoid leucosis, but is rather an atypical erythro or myeloid leucosis.

If, in fact, the visceral form of fowl paralysis (visceral lymphomatosis) and lymphoid leucosis are distinct conditions with a different aetiology and mode of transmission, the controversy is resolved, since it is evident that the rival schools are reporting results for the particular condition with which they happen to be working. It is the purpose of this paper to present evidence that this may be the case, and that the various forms of fowl paralysis should be considered apart from the leucotic complex.

It is obviously not possible in the short time at our disposal to go fully into the pros and cons of this controversy, but the reader may find most of the necessary data in Biester & Schwarte (1952) and in a paper by Campbell (1954).

Seasonal and Age Difference

Attention has already been drawn to the fact that the leucoses were known in Britain for a considerable time prior to the first recorded case of fowl paralysis. On the whole, fowl paralysis affects a younger age group of birds than leucosis, and in fact it shows a significant seasonal variation, which leucosis does not. The peak months for fowl paralysis are August to October, and since the majority of fowls are spring hatched, and the available evidence shows that the birds are infected during the first week or so in life, this peak gives us a guide to the incubation period before clinical paralysis or other associated lesions appear, *i.e.* about five to eight months. This is, in fact, the age when most cases occur, although there now seems to be a tendency for even younger birds to develop symptoms. Leucosis, on the other hand, usually affects an older age group, when the birds are already in full production.

Differences in Pathology

The leucoses are neoplastic conditions affecting haematopoietic tissue. As such, they are divisible into lymphoid, myeloid, and erythroleucosis. Each of these may be sub-divided into diffuse and discrete forms, in descending order of malignancy. In addition, the diffuse forms may show a true leucaemia, though this is usually terminal. The exception is erythroleucosis, which is nearly always leucaemic and only rarely occurs as aleucaemic diffuse or discrete forms. These leucoses show all the accepted criteria of malignancy such as rapid proliferation, uniformity of cell type with undifferentiated or relatively immature cells, infiltrations and destruction of invaded tissues. In addition, at least two of the

leucoses, namely erythro and myeloid leucosis, are associated with a filterable agent or " oncogenic virus," whilst the third, or lymphoid leucosis, is only doubtfully so associated, as we have already seen.

In the neural form of fowl paralysis, the early lesion consists of oedema of the nerve, followed by an infiltration with typical chronic inflammatory cells, mainly mature lymphocytes and plasma cells, which may be so severe as to destroy all normal nerve structure. A somewhat similar process occurs in the development of the visceral lesions, although usually the earliest sign is a minute focus of fibrinous necrosis, followed by an infiltration with lymphocytes, plasma cells, and even cells of the granular series. At this stage the lesion closely resembles an early pullorum disease focus. The mature lymphocytes rapidly aggregate at the site and finally a " tumour " or rather, a lymphogranuloma, is produced. This lesion bears a gross resemblance to the lymphocytoma of discrete lymphoid leucosis, but does not typically behave in a malignant manner. Figs. 1 to 8 compare fowl paralysis lesions with similar organs affected by lymphoid leucosis.

Although these lymphogranulomatous " tumours " are usually associated with nerve lesions in any particular case, they occasionally occur in the apparent absence of neural infiltration. That these cases can with practice be differentiated from lymphoid leucosis tumours is well shown in a study by J. E. Wilson and myself comparing results obtained from independent diagnosis based on gross *post-mortem* findings with the results of histopathological examinations. In a large series of *post-mortem* examinations, several such cases have been found, with excellent agreement as to their nature. It would of course be extraordinary if an exception to a general rule never occurred in any biological study, and on rare occasions a malignant transformation of fowl paralysis lesions has been observed, terminating in frank lymphosarcomata. This is not unexpected, however, being simply an example of the occasional conversion of a chronic inflammatory lesion to a true neoplasm. The fundamental difference in their beginnings, however, probably constitutes the best evidence for separation of fowl paralysis and associated conditions from the leucoses, which are neoplastic diseases from the outset.

Other points of difference may briefly be detailed, such as the absence of marrow or blood changes in fowl paralysis, or the practically invariable absence of nerve involvement in the leucoses. The production of fowl paralysis following inoculation with material from a leucosis case has occasionally been reported, also the converse, but the possibility of spontaneous disease confusing the issue was not by any means eliminated. The great majority of workers, and the writer is one, have never succeeded in transmitting fowl paralysis in any form following the injection of many different tissues or filtrates derived therefrom into fowls of widely different ages, whereas it is common experience that the leucoses may be transmitted in this way, although most would agree that lymphoid leucosis is difficult to transmit by cell-free material.

To sum up. It is held that fowl paralysis is typically a chronic disease associated with inflammatory infiltrations in the nerves and viscera, and a progressive though not neoplastic accumulation of cells of the lymphoid series which eventually produce tumour-like lymphogranulomata. There is no blood or marrow involvement, and no conclusive evidence that leucosis has ever resulted following attempted transmission experiments with fowl paralysis material or vice versa. The epidemiology of fowl paralysis shows a number of significant differences when compared to the leucoses, which are true neoplastic diseases of haemotopoietic tissue.

References

BIESTER, H. E., & SCHWARTE, L. S. (1952). Diseases of Poultry. Iowa State College Press. 3rd Edn.
CAMPBELL, J. G. (1954). Tenth World's Poultry Congress. Sect. C. 1.
GALLOWAY, I. A. (1929). *Proc. Roy. Soc. Med.* xxii, 1167.
WILSON, J. E., & CAMPBELL, J. G. (1956). Unpublished data.

The Practical Importance of Leucosis and Fowl Paralysis

BY

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I WISH to make it clear in my opening remarks that I shall not attempt to differentiate between leucosis and neural lymphomatosis (fowl paralysis) when discussing the importance of these diseases to the poultry industry. In doing so I shall not be creating a precedent, because it has become customary to speak of these conditions under a single heading. It is impossible, except on general lines, to assess the relative importance of the leucotic diseases as compared with lymphomatosis (fowl paralysis). I propose to deal first with the economic importance of these diseases as far as it is possible to estimate it.

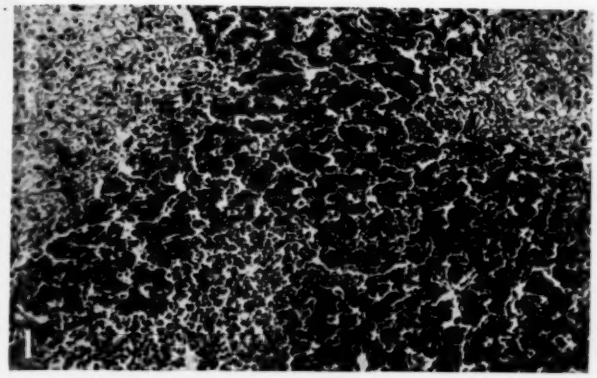
A. Economic Importance

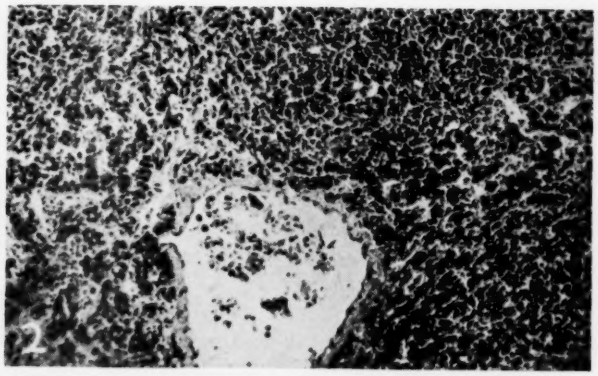
It is not easy to obtain a reliable and accurate estimate of the monetary loss from a single group of diseases. I propose to try to do so by reference to published figures of mortality at laying trials, and to the records of the diagnostic laboratories of the Ministry of Agriculture.

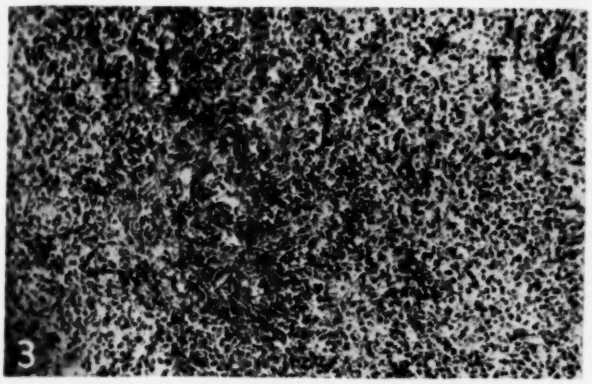
a. *Laying Trials*

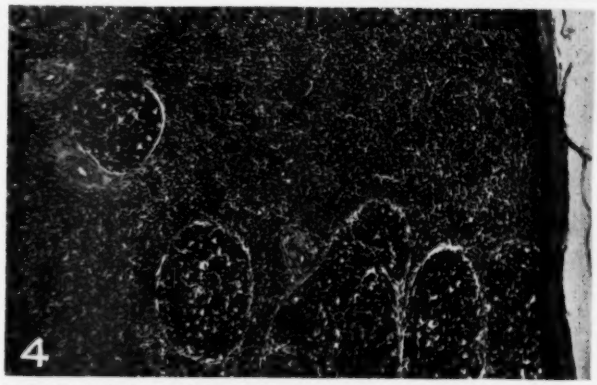
Dudley, Dobson & Gordon (1941), analysed the *post-mortem* records of both county and open laying trials in England and Wales, 1930-37. The total mortality increased from 6.3 per cent. to 17.3 per cent. during these years, but the average incidence of lymphomatosis and leucosis was 32.9 per cent. of total deaths at county trials and 23.7 per cent. at national trials. These figures were based on a 48-week period during the pullet laying year.

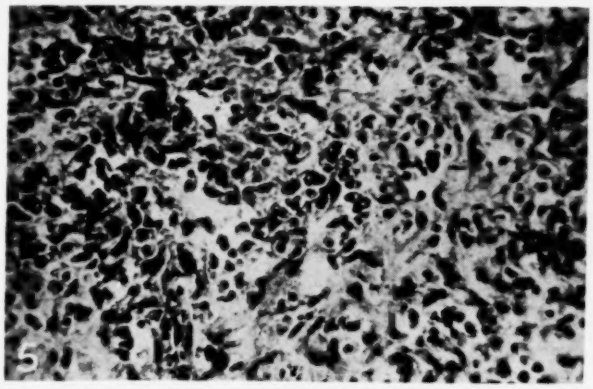
The over-all mortality at the national laying trials carrying on since the war has fallen to between 8 and 9 per cent., but the percentage of the total mortality











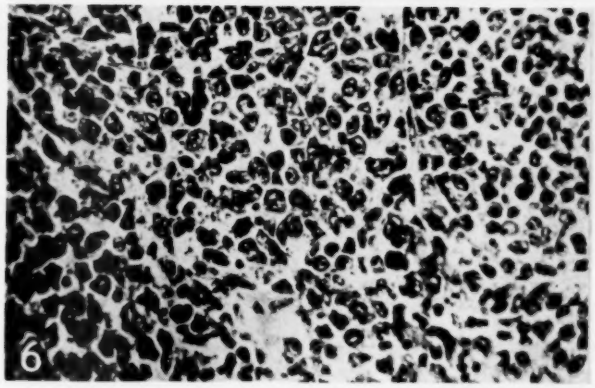


FIG. 1.—Early visceral fowl paralysis, liver. Two regions of fibrinous necrosis in upper part of photograph, and infiltration of lymphoid cells into an older lesion below. H. & E. × 130.

FIG. 2.—Diffuse lymphoid leucosis, liver. Note terminal leucaemia. H.&E. × 130.

FIG. 3.—Early fowl paralysis lesion, spleen. H. & E. × 130.

FIG. 4.—Early lymphoid leucosis, spleen. Explosive multi-centric growth in follicles. H. & E. × 110.

FIG. 5.—Typical early lymphogranuloma, fowl paralysis, ovary. H.&E. × 325.

FIG. 6.—Lymphoid leucosis, ovary. H. & E. × 325.

FIG. 7.—Lymphogranulomatous deposits on serosa of intestine. There is no tendency to invade the underlying musculature. H. & E. × 110.

FIG. 8.—Lymphoid leucosis. Tumour deposits on serosa of gut, showing invasion of muscle coats. H. & E. × 110.

attributed to leucosis and lymphomatosis in recent years has remained very close to that of the pre-war years. The average mortality from leucosis at the Yorkshire Federation Laying Trial, 1950-55 has been 19.8 per cent. The same figure at the National Laying Test for 1950-53 was 30 per cent. There have been no County Laying Trials since the war.

The figures from the Poultry Production and Progeny Trials in Lancashire are even more disturbing, since here the records are kept of mortality from day old to the end of the first laying season, *i.e.* 18 months old. For the years 1950-53 leucosis accounted for 35.7, 27.6, 41.0 and 53.0 per cent. respectively of a total average mortality of 21.7 per cent.

b. *Post-mortem Records*

The figures from the *post-mortem* records of the Ministry's laboratories at Weybridge and Lasswade, present a similar picture of the incidence of these diseases. The examinations include poultry from all branches of the industry: from laying trials, pedigree and commercial breeding flocks, smallholders, and domestic poultry keepers. An average of 23.4 per cent. of the 9,000 or so adult fowls received at Weybridge every year for the past four years have died from leucosis or lymphomatosis, and at Lasswade the figure is 24 per cent. of approximately 6,000 fowls examined every year.

c. *The Estimated Cost of the Mortality*

Coles (1955) estimated that the annual loss from mortality in the present poultry population of the United Kingdom with an annual output of approximately £150,000,000, is about £10,000,000. He suggests that the death rate reduces the potential egg output by another £20,000,000 and that the actual loss of potential income altogether is about £30,000,000 annually.

On the figures already given, based on laying trials and *post-mortem* records, *i.e.* about 25 per cent., it is clear that approximately £7,000,000 to £8,000,000 per year are lost in the poultry industry owing to the ravages of leucosis and lymphomatosis.

The figures for the production and progeny trials suggest that the actual percentage of deaths attributable to leucosis may well be in excess of 25 per cent. of the total mortality. Furthermore, Coles published in the same paper records of the mortality in a flock of White Leghorns over a period of four seasons, each of 78 weeks. 4,181 pullet chicks were hatched and 22.0 per cent. died before they were 18 months old; 10.18 per cent. of these deaths over the four seasons were due to leucosis, *i.e.* about half the total losses. On Coles' figures, the monetary loss to the industry from leucosis every year could be in the region of £15,000,000.

As a postscript to this estimate of the economic loss due to leucosis and fowl paralysis, it is interesting to note that according to Lord Rothschild, in an address to the Farmers Club in December, 1955, the total expenditure in the United Kingdom on poultry research for 1954-55 was about £122,000; this figure included money spent on research on poultry husbandry and nutrition as well as disease.

Before going on to discuss the practical importance of leucosis and fowl paralysis or, rather, the impact of these diseases on everyday life in the poultry industry, it might be useful to indicate a few of the facts revealed by research workers during the last 15 years or so.

B. Epidemiology of the Disease Complex

a. *Cause*

The consensus of opinion is that the causal agents of leucosis and lymphomatosis are viruses, but little is known of their relationship to one another. These agents are probably ubiquitous and inescapable except by rigid isolation.

b. *Age Susceptibility*

Most workers accept the view that the baby chick is very susceptible to infection for the first three to four weeks of its life. Resistance increases with age until, in the case of the adult, it is virtually impossible to obtain infection even by artificial injection. In practice, infection is thought to take place during this early, susceptible period, although evidence may not be forthcoming for several months. Clinical cases of neural lymphomatosis frequently occur in an infected flock two or three months before those of the leucosis. Neural lymphomatosis often causes serious loss in chickens two to three months old, but leucosis is seldom evident before five to six months.

c. *Existence of Carriers*

It is generally accepted that symptomless "carriers" are present in most flocks and that they are capable of infecting susceptible baby chicks (Burmester, Gentry & Waters, 1954).

d. *Egg Transmission*

Most people accept the probability that egg transmission of certain of these diseases does occur, but the importance of this factor in the spread of the disease is still hotly debated (Cole, 1949; Cole & Hutt, 1951). The evidence of egg transmission of certain types of leucosis (East Lansing workers—Cottral, Burmester & Waters, 1954) is irrefutable, but there is little or no evidence of egg transmission of neural lymphomatosis.

e. *Genetical Susceptibility*

Genetic differences in susceptibility among families were observed more than 20 years ago (Kennard, 1933). Resistant and susceptible strains of fowls have been established. The work of Hutt & Cole (1944) at Cornell is outstanding on this subject. They claim that it is a practical policy for the ordinary poultry farmer to develop resistant strains.

f. *Treatment*

Drug therapy including use of antibiotics and vitamins is regarded as useless. Ailing fowls should be killed, but the carcases are suitable for table if culled before loss of condition is advanced.

Chubb & Laursen (1954) reported that aminopterin and a-methopterin appeared to check the growth of

a transplantable lymphoid tumour, but the practical value of this work seems doubtful, particularly as these substances were found to be very toxic.

g. *Vaccination*

Burmester (1955) at East Lansing has reported experiments involving 14 immunised birds and 300 test chicks, which indicated that the virus of visceral lymphomatosis is capable of inducing antibodies in adult fowls, and that these antibodies are transmitted to the chick via the egg, conferring to the chick a significant immunity to challenge inoculation. The practical importance of this work is not great as far as can be seen at the moment, *e.g.*, Burmester was working with only one type of leucosis.

It can be seen readily from this brief review that leucosis and neural lymphomatosis are bound to have a profound impact on day-to-day management in all sections of the poultry industry.

C. Practical Importance on the Farm

a. *The Commercial Egg-producer or Rearer*

The rearer of table chickens is unlikely to experience serious losses from these diseases, although we have seen sporadic cases of neural lymphomatosis in chicks as young as three to four weeks, and mortality in chickens two months old is not uncommon. Generally, however, losses are seldom serious before a flock of table poultry is ready for market.

The rearer and owner of pullets for commercial egg production may experience a serious setback from neural lymphomatosis among his young chickens before maturity, and again from leucosis after production has started.

The severity of the losses in a flock is thought to depend on the degree of infection to which the chicks were exposed during early rearing and to the inherent resistance or lack of it in the stock. Some people believe a " trigger factor " may operate in this disease; it is certainly true that environment during rearing does seem to play a part in some outbreaks; for example, occasionally, serious loss occurs in one group of chickens and negligible loss in another, despite the fact that they were both hatched together and reared in contact for the first six to eight weeks before being sold to different owners.

The measures by which the incidence of leucosis and paralysis might be reduced in subsequent batches of chicks constitute a considerable problem to the average poultry owner. The work of Hutt & Cole (1944) at Cornell, and also that of Harris, Johnston & Wilson (1939, 1947) in Scotland has shown the value of isolation during the first susceptible weeks of the chick's life, and Hutt, in particular, has emphasised the importance of as great a distance as possible between the brooder houses and adult fowls, and also the use of clean ground for rearing. To apply this advice on the average farm presents great difficulties which are sometimes insuperable.

The work of Gordon & Coles (1955) at Houghton suggests that the practical application of " the rearing in isolation " recommendation may not always be as effective in reducing losses as its original sponsors had hoped.

b. *The Hatchery*

The hatchery owners are chiefly concerned in the problem of leucosis and paralysis because of the threats of legal actions from dissatisfied customers, whose chickens were brought from the hatchery at day old or, perhaps occasionally, as growers four to eight weeks old.

The experimental evidence of egg transmission of the virus causing one form of leucosis (reported as visceral lymphomatosis by the East Lansing workers) must be accepted; furthermore, field experiences both in America and this country, strongly suggest that egg transmission must play a part in the transmission of these diseases. But experimental proof that neural lymphomatosis is egg transmitted is lacking.

The East Lansing workers have also demonstrated incubator dissemination of their leucosis virus and infection by inhalation (Burmester & Gentry, 1954).

The position of the hatchery owner is, therefore, very difficult. He cannot be certain that the day-old chicks he supplies are free from these diseases, and yet, apart from maintaining efficient incubator hygiene, including fumigation of incubators, there is little that he can do about it.

c. *The Breeding Flock*

The owner of a breeding flock selling either day-old chicks himself, or supplying hatching eggs to a hatchery has much the same problem.

Kissling (1947) in America evolved a serological test for the detection of the " carrier " hen. It was a rapid plate agglutination test, using an antigen of stained canine lymphomyctes. Darcel (1950) working at Houghton, tried out this test, but found it too unreliable to justify use in the field. More recently another test has been worked out in America (Lescher & Burmester, 1955) based on the production by the virus of an enzyme in the blood plasma which reacts with adenosine triphosphate; there is no evidence yet of its value.

In any case, the possibility of eradicating leucosis and lymphomatosis, in all their various manifestations, seems unlikely. It is probable that the diseases are present in most of the chickens in a group that has been exposed and, if an efficient test were devised, it would disqualify almost every bird in a breeding pen.

The owner of a breeding flock must, therefore, face the fact that he cannot guarantee that his eggs and day-old chicks may not be infected, but, as in the case of the hatchery, he cannot fairly be expected to accept liability for leucosis and neural lymphomatosis, which may occur subsequently in stock he supplied as day olds.

In the case of sale of growing chickens, however, it might be argued that a heavy incidence of leucosis was the result of exposure during the early weeks of rearing. It follows, therefore, that day-old chicks, reared by the breeder or by a hatchery for sale as growing chickens or pullets, must be isolated, as far as possible, from adult fowls.

The development of strains genetically resistant to leucosis and lymphomatosis has been studied over many years by Waters & Pricket (1944) and by Hutt

& Cole (1947, 1948) in America. It is their view that these diseases can be controlled by careful breeding to eliminate susceptible families and to accumulate genes for resistance, with a view to producing strong stock which can withstand ordinary exposure to the disease. Many of the foremost poultry breeders in this country state that they seldom experience trouble in their own flocks from leucosis, nor do they receive complaints from customers who purchase their stock. Few of these men have followed a deliberate breeding policy in regard to leucosis, but all of them have probably practised selective breeding for many years, including an attempt to eliminate families in which mortality has been high. It may be, that without deliberately trying to do so, they have succeeded in controlling leucosis on their farms.

On the other hand, Coles (1954), after five years' experience in trying to establish genetically resistant strains on an experimental farm, is of the opinion that the substantial financial expenditure involved is beyond the means of most practical farmers. He points out that it is necessary to retain a susceptible line to ensure that the infection still exists, and that the resistant lines are genuinely resistant. In a second paper (1955), Coles presented evidence from the same experimental flock to suggest that a number of dams who produced families free from leucosis when mated as pullets, produced, when mated as hens, a number of daughters succumbing to the disease. He therefore questions the policy of advising poultry keepers to breed from hens, rather than from pullets, unless a fair measure of genetic resistance already exists in the flock. Coles also drew attention to the fact that fertility and hatchability was higher among the dams whose progeny succumbed most to the disease; his records over several years revealed a close correlation between high hatchability and the incidence of leucosis in the progeny of dams evincing high hatchability. Coles concludes that breeding for resistance may be a more complex subject than is generally thought to be the case. In view of Coles' observations on breeding resistant strains, I think we should avoid being too dogmatic when advising farmers on the genetic control of leucosis, but, nevertheless, the experience of many of our own long-established breeders of poultry does suggest that this is one of the keys to the problem.

Furthermore, Hutt & Cole (1955) in a recent communication express strong disagreement with Coles' views on an apparent relationship between susceptibility to the disease and efficient reproduction, maintaining that under the conditions of his experiments such a conclusion was not justified. The American authors state that from their own records, it is clear that resistance and susceptibility to lymphomatosis in the progeny are not related to the efficiency of reproduction in the dam.

References

BURMESTER, B. R. (1955). *Proc. Soc. Exp. Biol. N.Y.* **88.** 153.

———, GENTRY, R. F., & WATERS, N. F. (1954). *Poult. Sci.* **33.** 1046.

———, ———, & ———. (1955). *Ibid.* **34.** 609.

———, & ———. (1954). *Proc. 91st Ann. Meet. A.V.M.A.* 311.

CHUBB, L. G., & LAURSEN, A. L. (1954). *Brit. J. Pharm. and Chem.* **9.** 4, 419.

COLE, R. K. (1949). *Poult. Sci.* **28.** 31.

———, & HUTT, F. B. (1951). *Ibid.* **30.** 205.

COLES, R. (1954). *Ibid.* **33.** 350.

———. (1955). *Ibid.* **34.** 312.

———. (1955). *Brit. Vet. J.* **111.** 235.

COTTRAL, G. E., BURMESTER, B. R., & WATERS, N. F. (1954). *Poult. Sci.* **33.** 1174.

DARCEL, C. LE Q. (1950). *Vet. Rec.* **62.** 621.

DUDLEY, F. J., DOBSON, N., & GORDON, R. F. (1941). *Ibid.* **53.** 323.

GORDON, R. F., COLES, R., & STACEY, C. G. (1955). *Ibid.* **67.** 297.

HARRIS, S. T., JOHNSTON, J. W., & MITCHELL, S. G. (1947). *Vet. J.* **103.** 301.

HUTT, F. B., COLE, R. K., BALL, M., BRUCKNER, J. H., & BALL, R. F. (1944). *Poult. Sci.* **23.** 396.

———, & ———. (1947). *Science* **106.** 379.

———, & ———. (1948). *Proc. 8th World's Poult. Cong.* 719.

———, & ———. (1955). *Nature.* **176.** 1,178.

JOHNSTON, J. W., & WILSON, J. E. (1939). *Vet. J.* **95.** 484.

KENNARD, D. C. (1933). *Poult. Sci.* **12.** 335.

KISSLING, R. E. (1947). *Ibid.* **26.** 74.

LESCHER, S., & BURMESTER, B. R. (1955). *World's Poult. Sci. J.* **11.** 30.

WATERS, N. F., & PRICKETT, C. V. (1944). *Poult. Sci.* **23.** 321.

REPORT

The O.E.E.C. European Grassland Conference, held in Paris in 1954, has just published its proceedings in the form of a substantial book of some 400 pages. It covers a remarkably wide range of discussions of grassland production and conservation the world over, with special reference to problems of individual countries. The proceedings are indicative of the close application of scientific thought to the problems of production of foods of animal origin from the resources available in individual countries to meet the modern trend to higher standards of living.

Outstanding contributions in many fields were made by delegates to the Conference. It does seem strange, however, that when discussions of an international character concerned with grassland production and management are held, that there is no indication of anyone with a veterinary degree having attended from the United Kingdom, or, apparently from other countries. The work of Dr. Davies and Mr. Williams in the ecology and physiology of fodder plants was notable in its section; and that of Dr. De Vries and M. Boer of the Netherlands, and M. Andres of Belgium, appear to have given very interesting results in practice and may have considerable effect on future grassland production in this country. It is most interesting to find that the section dealing with grassland passed the following resolution: " That at the next conference a special section will be set up to study these problems and the behaviour of grazing animals." " These problems " refers to the management of grassland particularly in intensive grassland farming. It would seem that the veterinary profession, internationally, would have much to contribute to such a discussion.

The Continuation of Infection with *Streptococcus agalactiae* through the Dry Period in the Cow

BY

D. G. HOWELL*

Agricultural Research Council, Field Station, Compton

IT is generally agreed that *Streptococcus agalactiae* can persist in the bovine udder through a dry period of normal duration. During the course of studies on the pathogenesis of *Str. agalactiae*, being carried out at this Field Station, seven cows that had been experimentally infected in one quarter by the introduction of *Str. agalactiae* into the teat sinus, were dried off with infection still present. The infection had been present for periods varying respectively from 4 to 48 weeks. As shown in the Table they were dry for periods varying from 12 to 53 weeks. All of them yielded *Str. agalactiae* from the originally infected quarter immediately on coming into lactation. As will be seen the organisms were one of two different strains which were of different serological type. Strain S.13 belongs to Lancefields Type Ib and A100 to Lancefields Type II. The organisms recovered were of the same serological type as those present at drying off, and since they were only obtained from the original quarters it seems reasonably certain that infection was, in fact, carried through the dry period.

The cows in question were all dry for a very long time, and it is of note that all of them yielded organisms immediately on calving. In general farming practice, when cows are not dry for more than two months or so, it would seem likely that a cow dried off with *Str. agalactiae* in the udder would calve down with infection still present.

CONTINUATION OF INFECTION WITH *Streptococcus agalactiae* THROUGH THE DRY PERIOD IN THE COW

Cow number	Strain infected	Number of weeks organisms recovered in previous lactation	Number of weeks dry
1	A100	27	33
2	A100	4	53
3	A100	13	25
4	A100	43	20
5	A100	13	36
6	S13	48	12
7	S13	29	37

* Present address, Glaxo Laboratories, Greenford, Middlesex.

INVITATION FROM FRANCE

A French veterinary surgeon and his wife, living in Bordeaux, would be pleased to receive a boy or girl, aged about 16, to stay with them during the holidays.

Alternatively, they would welcome a young British veterinary graduate, man or woman.

Anyone who may be interested is asked to write to the General Secretary of the B.V.A.

Operation for the Retention of Prolapse Anus and Rectum after Reduction

BY

R. RICHARDSON

Chesterfield

PROLAPSE of the anus and rectum in the sow is frequently encountered in general practice. The condition usually presents itself shortly after farrowing. The operations described in veterinary text-books for retention have never proved entirely satisfactory. Accordingly, when a new approach to the problem was suggested by a medical practitioner I was eager to give a trial in my practice to a modified version of the operation. As a result the following technique has been developed, and 23 successive cases have given excellent results.

Restraint

This is accomplished by means of a noose over the upper jaw firmly anchored behind the canine teeth, the free end of the rope being securely tethered in front of the sow. An assistant is required to hold the sow's tail, and to push her firmly against a wall.

Operation

The peroneum, cleansed and suitably disinfected, is anaesthetised by infiltration with Xylotox. An eyeless curved cutting needle, with surgical steel wire attached (No. 32 B.W.G.) is used. The needle is inserted approximately half an inch from the border of the anus, and is guided through the tissues deep to the internal anal sphincter muscle to circumscribe the anus. This can usually be achieved in two arcs, the final point of emergence of the needle being close to the point of initial insertion.

The prolapse is reduced, and the steel wire is pulled sufficiently tight to form an orifice in the anus sufficient for the passage of faeces (to admit two fingers). A triple throw surgeon's knot is made, and the ends of the wire cut short and turned in. The skin is nicked below the knot which is then buried. No further treatment is required. The wire is left *in situ*.

In four cases in piglets, where 20-day chromic catgut of suitable thickness replaced the steel wire, excellent results have been achieved.

Acknowledgments.—Gratitude is expressed to Dr. R. H. Richardson, who described the operation as applied to human surgery.

INCREASES OF LIVESTOCK

The agricultural census taken early in June has shown that compared with last year there has been an increase of 3 per cent. in the total of cattle and calves. Sheep are up by 3 per cent., including an additional 319,000 ewes kept for breeding. Total pigs have fallen by 255,000 (5 per cent.), but gilts-in-pig have increased again after the sharp fall last year. The total of females for breeding shows a slight increase. Fowls have increased by 4,500,000, including an extra 800,000 turkeys.

Current Literature

ABSTRACTS

Role of Vitamin E in Poultry Nutrition and Disease. AMES, S. R. (1956). *Poult. Sci.* **35.** 145-59.

The author reviews the various functions of vitamin E together with the various symptoms of vitamin E deficiency in poultry. The latter include nutritional encephalomalacia ("crazy chick disease") and exudative diathesis in young chicks, muscular dystrophy in ducks, gizzard erosion in chicks and turkey poults, an enlarged hock disorder in turkey poults and a similar hock condition in older turkeys.

The vitamin E content of various foods is discussed. Young pasture, dried grass and dried lucerne are rich in vitamin E, but mature pasture is a poor source. It is difficult to judge the adequacy of any given diet since the vitamin E tends to be lost by oxidation during storage. It is also found that birds may be unable to utilise all of the vitamin E shown to be present on chemical analysis.

The author quotes 90 references.

E.A.G.

Sources of Tuberculous Infection in an Area containing Bovine Tuberculosis. ZWANENBERG, D. F. van, STEWART, C. J., HARDING, K. M., & GRAY, S. T. G. (1956). *Brit. Med. J.* June 23rd, 1956. 1,464-6.

In areas where the milk is known to be "safe" infection of tuberculin-positive school entrants can often be traced to a human source. On occasion tuberculin-testing of school children has been done in a limited area to estimate the damage done by a tuberculous cow. The present report deals with an outbreak of bovine tuberculosis in children in a small town in the Eastern Counties which was, it is claimed, traced to its source. The location was a small market town with a population of 7,400.

During four months in 1954, five cases of tuberculous cervical glands and one case of tuberculous peritonitis were discovered among children in this town. In each case there was a history that the child had been drinking raw milk from one particular dairy. At that time milk from all four dairies in the town had been satisfactory to bacteriological and statutory tests for a number of years. It was therefore decided to do a tuberculin survey of the school children of this town. Of 1,063 school children who were tuberculin-tested 260 were found to be positive. Of these 37 had a definite history of contact with a known case of tuberculosis, a further 25 had lived most of their lives outside the town and had drunk raw milk direct from a farm at some time. The remaining 198 children had no history of contact, and except for short periods had always had their milk supply from local dairies.

These children were listed according to the dairy which supplied them. It was found that of the 40 positive children under seven years, 31 had milk from dairy A. When the number of tuberculin-positive children was related to the quantity of milk supplied by each dairy it became clear a co-relation would be established between the incidence of tuberculin-positive children and dairy A and that this co-relation was nearly complete in respect of children under seven.

The history of the four dairies appears to substantiate the co-relation established by the tuberculin survey.

Dairy A. Milk sold by this retailer was the produce of his own farm. Undesignated milk was sold until the summer of 1954, when he made application for a T.T. licence. His herd was examined by the divisional veterinary officer, who found 19 out of 24 cows reacted to the intradermal tuberculin test.

Dairy B. has for the past four years sold T.T. milk only. It previously sold undesignated milk.

Dairy C. has sold only pasteurised milk for the last 19 years.

Dairy D. has sold only pasteurised milk for the last four and a half years. Previously sold T.T. and undesignated milk.

During the survey 12 further cases of cervical glands were found; the first 11 cases were supplied by dairy A.

This article concludes with a reference to possible danger of infection even when milk is pasteurised or from T.T. Herds. "Although there is control over the milk and cattle there is no direct control over those who attend cattle or 'handle' the milk. There are records of severe and sudden epidemics of bovine tuberculosis in cattle caused by an attendant having bovine tuberculosis of the lungs. It also is quite possible for a milk-handler with pulmonary tuberculosis to infect the milk direct." G.F.B.

The Relationship Between Particle Size and Anthelmintic Efficiency of Phenothiazine. DOUGLAS, J. R., BAKER, N. F., & LONGHURST, W. M. (1956). *Am. J. vet. Sci.* **17.** 318-23.

The authors state that a single dose of 25 grammes of a phenothiazine preparation with a small particle size (mean particle diameter between one and two μ) was about four times as effective in killing nematodes in the abomasum and small intestine of lambs as the same dose of a coarse preparation, with particles larger than 140μ. It was also about one-and-a-half times as effective in inhibiting the egg production of the nematodes. Similar results were obtained in mice experimentally infected with *Nematospiroides dubius*.

They suggest that further work is necessary to establish the optimum size of phenothiazine particle, which may differ according to the use for which the drug is intended. E.A.G.

The Removal of Chicken Tapeworms by Di-N-Butyl Tin Dilaurate. EDGAR, S. A. (1956). *Poult. Sci.* **35.** 64-73.

Di-n-butyl tin dilaurate was found to be highly effective against a number of poultry tapeworms—namely *Raillietina cesticillus, R. tetragona, Choanotaenia infundibulum, Davainea proglottina, Hymenolepis carioca* and *Amoebotaenia sphenoides*—when used for the treatment of field infected hens, most of which carried a mixed infestation. The drug was

(*Continued at foot of col. 1, page 534*)

Hand-rearing Badger Cubs

BY

L. BRIAN NETTLETON

Royal Veterinary College, Streatley

ON March 26th this year, while seeing practice in Yorkshire, I heard that a local game-keeper was going badger-digging the following day. As I am just as interested in the welfare of wild mammals as that of domestic species, I had a day's leave, with the result that both the badgers and I benefited. Three young cubs were saved, and I subsequently learned a great deal of badger temperament and behaviour by taking them off with me and rearing them.

The cubs were about 10 days old. Their eyes were still closed, and their wrinkled skin was covered only with a fine grey down and a vast number of fleas (*P. melis*). They were also hosts to a biting louse which I did not have identified, and to both larval and nymphal sheep ticks.

I had heard of unsuccessful attempts to rear both badger and other cubs on diluted cow's milk, and indeed I was advised to feed these cubs on such a mixture. However, I chose a proprietary food* which proved most suitable.

The cubs' first feed was not given until the evening, six hours after being unearthed. An improvised teat of bicycle valve tubing on a glass teat pipette was used. The feed was mixed and fed according to the instructions for two-week-old pups, and the cubs fed two-hourly that first evening, and three-hourly during the night. By the third day they had settled well to their new diet and were fed to appetite, four-hourly night and day, from a miniature baby's bottle. At each feed the hot-water bottle kept in their cardboard chick-box nest was refilled. This, I felt, was a most important point considering the constant and probably very high temperature within a badger's nesting chamber.

"Lactol" was their sole food, fed in increasing quantities and with longer intervals between feeds until, by their fourth week, 12 fluid ounces were being fed four times daily.

An important hint in rearing the cubs was given to me by a dog breeder. He termed it "Pottying," and it was carried out after feeds.

Essentially it was an imitation of the female actions in cleaning the cubs as they sucked. A cub was held in the left hand with the thumb over the neck and the forefinger within reach of the mouth. The first and second fingers of the right hand then tickled the perineal region. This immediately stimulated urination and a vigorous sucking of the left forefinger, accompanied by what I would describe as a delighted chattering.

It was not until eight weeks old that the cubs were completely weaned. They were gradually introduced to a thick oatmeal porridge made with milk, with glucose and cod-liver oil added, and dusted over with "Lactol." Soon afterwards raw liver was successfully introduced, and by the ninth week the cubs were supplementing their regular feeds with freshly killed birds, mice, slugs, and snails picked up on evening foraging trips.

Throughout the rearing of the cubs I tried to keep them under conditions as natural as possible. As they approached the age when they would normally be coming above ground (eight to nine weeks) they were first introduced to solid food and were encouraged to follow me from their "set" in a large dog kennel. As they gained confidence in the outside world I fed them as we went foraging in the neighbouring fields, by dropping pieces of food as I walked along. Thus, though their food was artificial, their mode of feeding was reasonably natural. Our foraging trips gradually lengthened until the cubs would walk with me as much as five miles during an evening.

On this programme of feeding and adequate attention to play and exercise the cubs have grown to weights ranging from 12 to 16 lb. at four months old. In this time my liking and admiration for the character of these elusive creatures has been confirmed. Better than this I feel that by showing off my adopted family I have done something to correct the deplorable attitude of too many individuals towards wild life.

* Shirley's "Lactol."

Abstracts.—*Continued.*

given either in the food for two to four days at the rate of 500 mg. per kilo of food, or else as a single dose of 125 mg. by mouth in capsule form. The treatment of experimentally infected birds gave similar results. In neither case were any toxic effects noted. A few tests were also made with other compounds.

It appears that *D. proglottina* is more difficult to eliminate than *R. ceticillus* or *C. infundibulum*.

Absorption of Vitamin A in Aqueous and Oil Solutions by Depleted New Hampshire Chicks. SQUIBB, R. L., BRAHAM, J. E., & SCRIMSHAW, N. S. (1956). *Poult. Sci.* **35.** 73-76.

When chicks that had been reared for five weeks on an experimental vitamin-A-deficient diet were given 7,500 I.U. of vitamin A in oil, either subcutaneous or intramuscular, the blood serum levels of vitamin A did not show a maximum response for seven days. In contrast to this, the administration of the same dose in aqueous solution, intramuscular, gave a maximum response within 24 hours.

Both oil and aqueous preparations gave a maximum response within 24 hours when given orally, but the authors point out that oral therapy may be less effective in diseased birds.

E.A.G.

News and Comment

THE CARIBBEAN VETERINARY CONGRESS

The second Veterinary Congress organised by the British Caribbean Veterinary Association opened in Kingston, Jamaica, on July 30th, and continued until August 8th.

Speaking at the opening ceremony, the Minister of Agriculture, the Hon. William Seivright, referred to the formation of the Caribbean Veterinary Association in 1946 and to the gradual strengthening of the veterinary services in the island. He said he thought this conference had special significance at a time when federation in the West Indies was in everybody's mind, and he referred to the recent introduction of legislation requiring the registration of veterinarians in Jamaica. He expressed his Government's welcome to the invited speakers who had come from Britain, the United States, and Canada, and also to the many veterinarians who had come from other parts of the Caribbean.

A reply to the Minister on behalf of the overseas delegates was made by Dr. E. L. Taylor.

The Congress had a full programme including sessions on nutritional deficiencies of farm animals, on bovine infertility problems, small-animal surgery and medicine, and parasitology. The formal sessions were interspersed with visits to Government experimental farms, to the Denbigh Agricultural Show, and with various social functions.

The Congress was, unfortunately, marred by the sudden and regrettable illness of Professor Weipers, who was to have presented the papers on small-animal surgery and medicine. Professor Weipers was taken seriously ill within a few days of reaching the island, but we are glad to hear that he is now progressing favourably. He will, however, need a long period of rest and recuperation. We extend to him our warmest wishes for a speedy recovery, and to Mrs. Weipers our sympathy for the anxious time through which she has passed. Mrs. Weipers who, fortunately, was with her husband when he was taken ill, presented his papers to the Congress.

The Congress has been well attended by delegates from Curaçao, Salvador, Guadeloupe, French Guiana, and Venezuela, as well as by representatives of the veterinary services of most of the British Caribbean islands.

Visiting speakers included Professor Robertson, whose paper was "Nutritional Deficiencies in Farm Animals," Dr. E. L. Taylor, "The Borderline Between Health and Parasitic Diseases," and Mr. H. E. Harbour, "Parasites and their Control." Dr. S. J. Roberts, head of the Ambulatory Clinic, Cornell University, spoke on "Ovarian Dysfunction in the Bovine," and Dr. J. A. Henderson, head of the Department of Medicine and Surgery, Ontario Veterinary College, spoke on "Diagnosis and Control of Infectious Diseases Causing Infertility in Cattle."

We hope to review the Congress in greater detail in a forthcoming issue.

THE PROFESSOR G. H. WOOLDRIDGE PORTRAIT FUND

At the Annual General Meeting of the Royal College, a number of tributes were paid to Professor Wooldridge, both by fellow members of the College Council and from the body of the hall. We feel that Professor Wooldridge's friends everywhere, and the profession generally, would wish to read the encomiums passed upon this great veterinarian, and they are, accordingly, published below.

The President said:

"To-day's meeting marks the end of an epoch in so far as it is the last meeting at which we shall have the advantage of the guidance and help of that great figure, Professor Wooldridge.

"His record is a wonderful one, not only in length of service but also by his personal devotion to the work of this Royal College. His attainments academic and professional, are marked and high, but I feel that his greatest attainment is the way in which by his courtesy, his simplicity and directness of outlook, he has entwined himself into our hearts. We thank you, Professor Wooldridge, for all you have done."

Mr. R. E. Glover: "May I be allowed to associate myself with the very moving words which you, Sir, have said about our colleague, Professor Wooldridge.

"Like yourself, I know that the words which you have spoken represent the views not only of this Council, but of the profession as a whole, and it is indeed an irreparable loss to the Council that Professor Wooldridge decided he would retire from our midst. I feel that as 'father' of the Council—and I use that word advisedly—sitting as he has done in what I regard as the place of honour, we have benefited for many years by his wise and considered views on all matters concerning the profession, and we shall miss that guidance in future.

"I think that one of the ways in which Professor Wooldridge has helped this Council to a very great extent is in the representation of our affairs on outside bodies. He has been appointed by us to more bodies, I believe, than any other member of the Council, and that I think is an indication of the high esteem in which he has been held by all of us. We all wish him the very best of fortune when he retires from this Council, and I think I can say that he will always be welcome at any function of the Royal College which is not concerned with the actual business of this Council."

Captain T. M. Mitchell: "I should also like to associate myself with the remarks of the President and those of Mr. Glover. I am in the unenviable position of having to accept the mantle which will be thrown off by Professor Wooldridge and I shall become the 'father' of this Council, although still a young man of course! I cannot hope to live up to the standards which Professor Wooldridge has set for the conduct of the 'father' of the Council. I can only emulate him in my own humble way.

"All I wish to say in addition to that which has already been said is that I hope that Professor Wooldridge continues to enjoy the excellent physical health which he now enjoys, except for the one disability which has compelled him to retire, and may we sincerely hope that that disability will not get any worse."

Miss J. O. Joshua: "As a very junior member of this Council I should also like to associate myself with the remarks which have already been made. Great tribute has been paid to Professor Wooldridge for his attainments in the profession, and I would only add one thing. Many generations of students hold him so much in their affection that he is known throughout the profession as 'Uncle George,' and as such we shall always think of him."

Dr. W. R. Wooldridge: "If I may, I should like to add my tribute to my real Uncle George. He has been a great stimulus to the younger people in the profession for very many years, but perhaps I have appreciated more than most, that what he has done for the profession and this Council has arisen out of love for the profession, and I think you can have no better example of that translation of love into the wider sphere than the life and work of Professor Wooldridge in the profession in general and in this Council in particular.

"Perhaps you would also allow me to add that I think we as a Council and as a profession owe something also to Mrs. Wooldridge, who has stood so steadfastly behind him throughout all his love and work for the profession."

Mr. H. Dawes (from the general body of members present): "I think it would be most fitting at a time such as this for those who are present here to-day as non-members of the Council, but who represent a very large body of the profession to be associated with the remarks of previous speakers in paying tribute to Professor Wooldridge.

"I have known Professor Wooldridge very much longer than I care to remember because time flies so quickly. I remember him very well even before I became an undergraduate at the Royal Veterinary College and he and Mrs. Wooldridge, that charming lady, his wife, were extremely good to me when I was at a very tender age. I think, therefore, that I can speak of knowledge of Professor Wooldridge at extreme length. Throughout the whole of that time, and particularly during the time I had the privilege of being one of his colleagues, he has been nothing but a source of encouragement and guidance.

"To the profession generally he has always been 'Uncle George,' and that goes both for graduates of the London School and graduates of every other school; and from the profession as a whole I should like to convey to Professor Wooldridge our hope that happiness and good health, qualified as it must be by what we know, will be his for many years to come. He is assured of this, that wherever he goes throughout the country he has only to call on any practitioner, any teacher or any research worker and he will be welcomed with open arms. It is given to very few men in our profession to reach such a stage of popularity and affection among his fellow members."

Mr. R. H. Smythe: "I think that I can claim to be Professor Wooldridge's first student. I was on the Committee of Students who received him when he came to the Royal Veterinary College, and I think I was the first student he ever ticked off thoroughly! I have appreciated him immensely ever since.

"When I was a student both he and Mrs. Wooldridge were extremely kind to me. I was one of those students who needed all the help he could get, and all through my professional career, if ever I have been in any difficulty Professor Wooldridge has always come to my aid. I only hope that any relief and rest he enjoys from carrying out those valuable duties which he has carried out on behalf of the College will put another ten years on to a very long life."

Further donations to the portrait fund have been made by the following:—

Col. G. Barnett, J. S. Blaxland, Esq., A. D. J. Brennan, Esq., J. Heath, Esq., W. C. Hesketh, Esq., H. E. Hornby, Esq., Major J. J. G. Keppel, Professor H. G. Lamont, Dr. M. S. Reichert, A. Sams, Esq., J. D. Shuja, Esq., A. L. C. Thorne, Esq., S. B. Vine, Esq., G. P. West, Esq., and D. A. J. Weedon, Esq.

Societies:

The Universities' Federation for Animal Welfare.

IN PARLIAMENT

Slaughterhouses and Knackers' Yards

MR. CHAPMAN (July 30th) asked the Minister of Agriculture, Fisheries and Food what improvements have now been carried out in the 15 horse slaughterhouses and 45 knackers' yards which in the early part of the year, were found to be unsatisfactory under the slaughter Regulations; and how the figures stand to-day.

THE JOINT PARLIAMENTARY SECRETARY TO THE MINISTRY OF AGRICULTURE, FISHERIES AND FOOD (MR. HARMAR NICHOLLS): Better arrangements have been made for feeding and watering horses, and in some cases there have been structural alterations. As a result, well over half these premises now comply with the Regulations. Four of the horse slaughterhouses and 15 of the knackers' yards have minor technical defects. In these and in the case of two others which officers of my Department consider more unsatisfactory, we are urging the local authorities to expedite action under their powers.

MR. CHAPMAN asked the Minister of Agriculture, Fisheries and Food in how many horse slaughterhouses and knackers' yards it is still the practice to kill horses in the sight of carcases.

MR. H. NICHOLLS: No incidents have been reported to my Department since the hon. Member asked a similar Question on April 12th.

MR. CHAPMAN: Is the Parliamentary Secretary not aware that we have been asking Questions on this subject for about 18 months, and still he tells us that the slaughterhouses and knackers' yards have not been cleared up, let alone at a satisfactory pace? If we put down some more Questions, will the hon. Gentleman be able to say that the matter will be cleared up by the autumn?

MR. NICHOLLS: The hon. Gentleman can be assured that his questioning has helped. We are trying to urge the local authorities who have failed in this matter to make use of their powers under the Regulations. The Answers which have already been given to the hon. Member show that we are getting local authorities to recognise the urgency of this matter, and that we are making progress. If the hon. Member knows of any specific cases, we will see that the attention of the local authority concerned is drawn to the matter.

MR. FELL: Is my hon. Friend aware that it is not very satisfactory to hear that only about half of the knackers' yards and slaughterhouses have come into compliance with the Regulations? My hon. Friend must be a bit tougher with them.

MR. NICHOLLS: My hon. Friend did not quite follow the purport of the Question and Answer. It was half of a specific number in an Answer which has already been given to the hon. Member. Half has already been cleared up. Most of the others have now got only technical defects which are being looked at, and we are down to two cases which we are looking at rather more urgently.

Experiments on Living Animals

MR. AWBERY (July 30th) asked the Secretary of State for the Home Department to what degree vivisection has increased in Great Britain during the past 10 years; how many dogs were experimented on, with and without an anaesthetic, in 1955; if he is aware that some national schools in medicine have abolished vivisection of dogs as unnecessary; and if he will take steps to do the same in this country.

MAJOR LLOYD-GEORGE: The number of experiments performed under the Cruelty to Animals Act, 1876, increased from rather less than 1,500,000 in 1946 to just under 2,500,000 in 1955. In 1955 the number of experiments performed on dogs without anaesthetic was 1,998, and with anaesthetic 1,894. The use of dogs for experiments, within the scope of the Act, is still considered to be essential in this country.

Horse Slaughterhouses (Inspections)

MR. CHAPMAN (July 31st) asked the Minister of Agriculture, Fisheries and Food how many visits have been made by his inspectors in the past three months to the nine horse slaughterhouses and knackers' yards which were found in the spring to be very unsatisfactory premises.

MR. AMORY: Nine visits have been made, one to each slaughterhouse.

Livestock (Exports)

MR. GIBSON-WATT (July 31st) asked the President of the Board of Trade the value of exports from the United Kingdom of pedigree livestock, under the various categories, for 1954 and 1955.

MR. P. THORNEYCROFT: Pedigree livestock are not distinguished as such in the Trade Accounts. With permission, I will circulate in the Official Report figures of exports of livestock classified as "for breeding."

MR. GIBSON-WATT: While thanking my right hon. Friend for that Answer, might I ask if, when he produces those figures, he can also give me some idea as to what percentage of the figures finds its way directly to the coffers of the Treasury as a result of these valuable exports?

MR. THORNEYCROFT: I can give the figures in sterling for the particular exports, and can show the comparison between the two years. Perhaps my hon. Friend will look at the figures, and if he wants any more information I will try to let him have it.

Following are the figures:—

United Kingdom exports	1954 £	1955 £
Animals for breeding		
Bulls	557,004	595,224
Cows	290,952	281,387
Calves	12,079	*
Sheep and lambs ...	44,225	
Swine	121,849	

* Not separately distinguished in 1955.

Meat Research (Technical Advisory Committee)

MR. CROUCH (July 31st) asked the Minister of Agriculture, Fisheries and Food if he will now announce the names of the members appointed to the Committee to look into the problems of the Meat and Livestock Industry.

MR. AMORY: Yes. I am glad to be able to inform the House that the following persons have agreed to act as members of the Technical Advisory Committee on Meat Research:—

Sir George Wilson (*Chairman*)
Professor Alex Robertson, Professor of Veterinary Hygiene and Preventive Medicine, University of Edinburgh.
The Earl of Antrim, Member of the Council of the Ulster Farmers' Union.
Mr. A. J. Austin, Butchery Manager, London Co-operative Society.
Mr. E. J. Baldwin, O.B.E., Ex-President of the Institute of meat.
Colonel L. B. Bennett, O.B.E., M.C., President of the Imported Meat Trade Association.
Dr. B. K. Blount, Department of Scientific and Industrial Research.
Mr. T. C. Brechin, Past President of the Scottish Federation of Retail Meat Trades' Associations.
Mr. E. C. Conn, Ministry of Agriculture for Northern Ireland.
Mr. H. R. Davidson, Farmer and agricultural journalist.
Mr. David Galloway, Vice-Chairman of Multiple Meat Traders' Association.
Mr. Frank Gerrard, National College of Food Technology,
Dr. John Hammond, C.B.E., Ministry of Agriculture, Fisheries and Food/Agricultural Research Council.
Mr. George Hedley, Ex-President, Scottish National Farmers' Union.
Mr. Maurice Passmore, Producer and meat expert.
Mr .J. H. Rodmell, President of the English Federation of Retail Meat Traders' Associations.
Mr. E. Roll, C.B., C.M.G., Ministry of Agriculture, Fisheries and Food.
Mr. T. J. Shaw, M.C., Managing Director (Marketing), Fatstock Marketing Corporation.
Mr. H. Shilleto, Chairman of Livestock and Wool Committee of the National Farmers' Union of England and Wales.
Mr. W. Shubert Jones, J.P., Chairman of the Welsh Committee of the National Farmers' Union of England and Wales.

Sir William Slater, K.B.E., Secretary of the Agricultural Research Council.

The Viscount Trenchard, M.C., Director, T. Wall and Sons Limited.

Mr. H. Whitby, Department of Agriculture for Scotland.

Mrs. Hilda Whitlow, Housewife and journalist.

Mr. James Williams, President of the Federation of Wholesale Fresh Meat Traders.

Professor H. G. Sanders and Dr. N. C. Wright, the Chief Scientific Advisers of the Ministry of Agriculture, Fisheries and Food for agriculture and food respectively, will assist the Committee as assessors.

THE AGRICULTURAL IMPROVEMENT COUNCIL

The Agricultural Improvement Council, which recently reached the end of a further three-year period of office, has now been reconstituted by the Minister of Agriculture. The Council was set up in 1941 to keep under review the progress of research, with a view to ensuring that promising results are applied as rapidly as possible to the problems of agriculture and horticulture and that these and any other new technical methods are incorporated into ordinary commercial practice. The Council also advises the Minister of Agriculture from time to time concerning agricultural and horticultural problems which appear to require scientific investigation, and generally suggests the lines on which a policy designed to raise the technical standard of agricultural and horticultural production can best be implemented.

The full membership of the reconstituted Council is as follows:—

Sir Alan Hitchman, K.C.B. (Chairman), Permanent Secretary, Ministry of Agriculture, Fisheries and Food; Professor Sir Frank Engledow, C.M.G., M.A., B.SC., F.R.S. (Vice-Chairman), Drapers' Professor of Agriculture, Cambridge University; Captain J. F. Bomford, M.C., Farmer and Market Gardener; Harold Collison, Esq., General Secretary, National Union of Agricultural Workers; H. R. Finn, Esq., Farmer, Nackington Farms, Canterbury, Kent: E. Gibby, Esq., Farmer, Upton Farm, Pembroke Dock; Professor K. Mather, C.B.E., D.SC., PH.D., F.R.S., Professor of Genetics, Birmingham University; J. W. S. Mount, Esq., Horticulturist and Farmer, Little Barton Farm, Canterbury, Kent; His Grace the Duke of Northumberland, Landowner and Farmer, Member of the Council of the Country Landowners' Association; H. J. Rathbone, Esq., F.L.A.S., Chartered Land Agent, Surveyor, and Valuer, Mold, Flintshire; F. Rayns, Esq., C.B.E., M.A., Director of the Norfolk Agricultural Station, Sprowston, Norwich; Professor Ellis J. Roberts, M SC., M.A., Professor of Agriculture, University College of North Wales; Professor Sir James Scott Watson, C.B.E., M.C., M.A., LL.D., Formerly Chief Scientific and Agricultural Adviser to the Minister of Agriculture and Fisheries; Sir William Slater, K.B.E., D.SC., F.R.I.C., Secretary, Agricultural Research Council; H. Cole Tinsley, Esq., M.B.E., Farmer, West Deeping Manor, Peterborough, Northants; Sir James Turner, B.SC., LL.D., President, National Farmers' Union; Professor T. Wallace, C.B.E., M.C., D.SC., F.R.I.C., F.R.S., V.M.H., Director, Long Ashton Fruit and Cider Research Station, Bristol; Professor E. G. White, D.SC., PH.D., F.R.C.V.S., Professor of Veterinary Preventive Medicine, Liverpool University.

THE COLONIAL VETERINARY SERVICE

Mr. D. H. L. Rollinson, PH.D., B.SC., M.R.C.V.S., Assistant Director of Veterinary Services (Research), Uganda, has been promoted Chief Veterinary Research Officer, Uganda. Mr. L. E. Stephen becomes Senior Scientific Officer, W.A.I.T.R., Nigeria.

There are the following new appointments: Mr. P. F. Byrne, Veterinary Officer, British Guiana; Mr. H. Steding-Jessen, Veterinary Officer, Gold Coast, and Mr. K. S. Thomson, Veterinary Officer, Tanganyika.

UNIVERSITY NEWS

Liverpool

Degree of B.V.Sc., Second Examination. Part III (Animal Management)

Adams, J.; Aspinall, Kathleen M.; Barnes, J. D. F.; Batty, A. F.; Bird, Christine M.; Butterfield, W. E.; Callender, D. E.; Capron, T. K.; Chesworth, A. T.; Edwards, J. D. D.; Fitton, J.; Fitton, M. W.; Foot, J. K.; Goulden, M. B.; Hanford, D. M.; Holt, H. J.; James, W.; Jenkins, J. T.; McMurtry, M. J.; Michael, H. P.; Miller, J. K.; Nyavor, A.; Pickering, P. L.; Pickup, Pamela; Quartey-Cofie, J. E.; Schofield, J. B.; Walker, J. M.; Weller, R. J.; Wild, G. S. P.; Worthington, P. T., and Zanetti, P. J.

B.V.A. NOVEMBER CONFERENCE

In connexion with the conference on Supplements and Additives in Animal Feeding-stuffs to be held on November 22nd and 23rd, we wish to advise our readers that particulars regarding application for tickets of admission will be published in the early autumn. The Conference is open to all members of the Association, but those wishing to attend are asked to notify Headquarters as admission will be by ticket only.

DEER POISONED IN RICHMOND PARK

Richmond Park officials and police officers are trying to find how four red deer and one fallow deer met their deaths from, it is suspected, arsenical poisoning.

Some years ago there was an outbreak of arsenical poisoning among the park deer, although none died. The source was then traced to the public golf course in the park, which had been sprayed with arsenic. The deer had wandered on to the course and eaten poisoned grass.

This could not be the cause of death on this occasion as arsenical sprays are no longer being used in the park. The body of one of the deer has been sent to a Ministry of Agriculture pathologist for *post-mortem* examination.

MYXOMATOSIS AWARD

Professor Armand Delille, the man who caused myxomatosis to spread throughout Europe by inoculating rabbits on his estate in 1952, has recently received a gold medal as a token of gratitude from the foresters and farmers of France.

At a ceremony near Chartres several speakers, including M. Bernard Dufay, honorary director-general of waters and forests, paid tribute to the professor's "courageous action." "Thanks to him," he said, "the forests of France have been enriched and crops are far less menaced."

Professor Delille then spoke of the need to continue the struggle against the rabbit pest.

WARBLE-FLY INFESTATION

Warble-fly infestation in cattle causes annual losses over the O.E.E.C. area as a whole to the value of some 30,000,000,000 francs. Most of these losses are in terms of milk and meat, but the value of hides and skins is also severely affected. As part of its programme for the improvement of animal health control in Western Europe, the European Productivity Agency of O.E.E.C. recently organised a meeting of experts to consider more effective means of combating this pest.

The purpose of the meeting was to enable all those actively concerned with the control of warble-fly infestation to discuss their experiences and to co-ordinate their future work, including enforcement measures and research projects. The main aspects considered were the incidence and economic effects of warble-fly infestation; practical methods of control and their application; control regulations and their enforcement; and publicity and education leading to voluntary action in this problem.

It was agreed that there existed a necessity for unified research programmes aimed at the discovery of a therapeutic agent which could be applied before the appearance of the warbles on the animals' backs, and the hope was expressed that the leather industry would continue to give its support to research work in this field.

MR. REGINALD W. HALL

Captain Ivor Hughes writes:—

Half a century ago I had the great good fortune to be sent to work for Reggie Hall, as he was known to a large number of his clients. Together with his young wife he was devoted to his work, having no time for social affairs, and frequently our day was made up of 18 hours, from farm to farm. His means of transport, two horses, used to be exchanged at mid-day so that his mount was always fresh.

Although kept hard at work with the larger animals, he would frequently do canine surgery for friends, of whom he had many. I pay humble tribute to his memory—a great man.

MR. THOMAS LISHMAN

Professor McCunn writes:—

Tom Lishman's many friends within and without the profession will have been grieved deeply, and experienced a great sense of loss, when they read of his passing. He qualified from the Camden School in July 1906 and soon afterwards joined the Royal Army Veterinary Corps in which sphere he spent his active professional life. A good soldier, he took every opportunity of advancing his knowledge, especially so by the experimental method.

His work, performed in India, on what I believe was his first tour abroad, solved the age-long speculation as to what happened when the superficial and deep flexor tendons and/or the suspensory ligament of the horse suffered severe damage amounting to rupture (*Vet. Rec.* **21.** 117). As one would expect of Lishman the text runs to very few words; but those words, and the magnificent photographs have ranked as "standard" since that time and been used to assist in solving many legal disputes.

He served throughout the first world war, and no doubt our contemporary, the *Journal of the Royal Army Veterinary Corps,* will record the details of his military career. When he retired from the Service he entered Grays Inn and qualified as a Barrister-at-Law.

It is probably from the legal angle that most of the younger generation knew him best. He was appointed lecturer in jurisprudence at the Royal Veterinary College. Like his old teacher, Professor James Macqueen, Lishman was not wasteful of words. He was able to translate the most abstruse legal verbiage into simple language that all could understand and follow. His lectures were cryptic, witty, and sprinkled with a subtle sarcasm. He must have read very widely in medico-legal matters for he was never at loss to quote some High Court or other legal ruling or precedent which had a bearing on the matter under discussion. This, of course, gave added interest and pleasure, and caused students to attend to learn instead of to collect the necessary mark in the register.

In the lecture room as well as in the Common Room Tom Lishman was a friendly man. He could enter into a philosophical discussion on almost any topic that might be raised. His nimble wit, and his twinkle of face and eye enabled him to oppose without inducing rancour.

He came and he went without any ostentation, and his passing was typical of the man. His friends had seen him make a wonderful recovery from a serious operation about a year ago, and we saw him at the R.A.V.C. Annual Dinner in June looking extemely well. We did not know of his death until the obsequies were over. This was, I believe, by his own wish and was a typical gesture.

PERSONAL

Death

BUCHANAN.—At Whiston Hospital, Liverpool, on July 28th, 1956, as the result of a motor accident, Frank, youngest son of Mr. and Mrs. J. M. Buchanan, M.R.C.V.S., 3, Morecambe Bank, Grange-over-Sands, and beloved husband of Dorothy Dyke.

COMING EVENTS

September

12th (Wed.). Meeting of the North Wales Division, B.V.A., at Leahurst Veterinary Field Station, 11.30 a.m.

16th to 22nd (Sun. to Sat.). 74th B.V.A. Annual Congress at Royal Leamington Spa.

November

22nd and 23rd (Thurs. and Fri.). B.V.A. Conference on Supplements and Additives in Animal Feeding-stuffs at the Victoria Halls, Bloomsbury Square, London, W.C.1.

ADDRESSES OF DISEASE INFECTED PREMISES

The list given below indicates, first the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

Anthrax

Ches. Thornfield, Newtown, Nantwich (July 31).

Cumberland. Town Head Farm, Great Salkeld, Penrith (July 31).

Lancs. Colbourne's Farm, School Lane, Lathom, Ormskirk (Aug. 2).

Salop. Wood End Hall Farm, Coton, Whitchurch (July 31).

Stirling. Townhead Farm, Fintry, Glasgow (July 31).

Worcs. Lodge Farm, Belbroughton, Stourbridge (July 31).

Fowl Pest

Lancs. Parkers-in-the-Fields, Charnock Richard, Chorley; Wood End, Marshes Lane, Mere Brow, Tarleton, Preston (July 31); 16, Westhoughton Road, Adlington, Chorley; Moorhey Farm, Little Hoole, Preston (Aug. 2).

Lincs. 51, Manor Street, Sleaford (July 31); Chapel Farm, Hawthorne Hill, Dogdyke; 63, Burton Road, Heckington, Sleaford (Aug. 2).

Swine Fever

Dorset. Holway Farm, Holway, Corton Denham, Sherbourne (July 31).

Essex. Street Farm, Ashen, Sudbury (July 31).

Hants. The Piggeries, Smallholding, Valley Road, Throop, Bournemouth (Aug. 3).

Herts. The Piggeries, West End Lane, Essendon, Hatfield (Aug. 2).

Lancs. Slate Pits Farm, Accrington (Aug. 1).

Lincs. Monksthorpe, Great Steeping, Spilsby (Aug. 2).

Midlothian. No. 13 Holding, Wester Hailes, Edinburgh (July 31).

Montgomery. Glanllyn, Leighton, Welshpool (Aug. 1).

Norfolk. Highview Farm, Swanton Morley, Dereham (July 31); Farrow's Farm, The Heywood, Diss (Aug. 1).

Salop. Piggery at Vicarage Buildings, Uppington, Wellington (Aug. 2).

Staffs. Piggeries at rear of 186, St. Paul's Road, Smethwick (Aug. 2).

Somerset. Little Pennard Farm, East Pennard, Shepton Mallet (July 31).

Worcs. Hollowfields Farm, Hanbury, Bromsgrove (Aug. 2).

CORRESPONDENCE

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

Torsion of the Bovine Abomasum

Sir,—I would be obliged if Mr. R. Richardson would give more precise detail on the physical endeavours necessary to correct this torsion. Being myself somewhat short of inches I find difficulty in reaching with the tips of the fingers of one hand the extreme limits of the bovine reticulum even approaching it in the direct line from flank and through the rumen. I have made several exploratory laparotomies on cattle in an attempt even to palpate the abomasum with the fingers of one arm fully entered into the abdominal cavity. I have always failed even to make contact. I would like to know how Mr. Richardson is able to enter both arms through a nine-inch flank incision and still be able to manipulate the abomasum with both hands. I have on occasion rectified a torsion of the large intestine from the left flank but only in small yearling animals.

Yours faithfully,

J. P. PICKERING.

Orchard House,
Priestpopple,
Hexham,
Northumberland.

July 27th, 1956.

ADVERTISER'S ANNOUNCEMENT

BURROUGHS WELLCOME & CO. announce the introduction of "Hepadis" brand Distemper, Hard Pad, and Contagious Hepatitis Antiserum.

This antiserum is prepared in dogs hyperimmunised against the viruses of distemper, hard pad disease and contagious hepatitis and also injected with mixed bacterial antigens. When used prophylactically, a dose of 5 c.c. is given subcutaneously, irrespective of the age or size of the dog. For therapeutic purposes, the antiserum is best given early in the course of the disease, in doses of 0.5 c.c. per lb. bodyweight.

It is issued in containers of 5 c.c. and 25 c.c.

DISEASES OF ANIMALS ACT, 1950—GREAT BRITAIN

Summary of Returns of Confirmed Outbreaks of Scheduled (Notifiable) Diseases

Period		Anthrax	Atrophic Rhinitis	Foot-and-Mouth	Fowl Pest	Sheep Scab	Swine Fever
1st to 15th July, 1956		19	2	—	31	—	34
Corresponding period in	1955	19	—	—	73	—	56
	1954	10	—	—	9	—	65
	1953	11	—	—	111	—	93
1st January to 15th July, 1956		1,037	8	13	354	—	421
Corresponding period in	1955	461	1	6	344	—	904
	1954	160	3	12	550	—	889
	1953	445	—	26	619	—	1,826

TUBERCULOSIS (ATTESTED HERDS) SCHEMES

The number of Attested Herds, *i.e.*, herds officially certified as free from Tuberculosis as at 30th June, 1956, was as follows :—

ENGLAND	WALES	SCOTLAND	TOTAL (GREAT BRITAIN)
87,442	35,007	37,672	160,121

TUBERCULOSIS ORDER OF 1938

The number of bovine animals slaughtered under the Tuberculosis Order of 1938 during the period 1st April to 30th June, 1956, was 297.